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☐	L12	424/130.1,141.1,142.1,143.1,145.1.CCLS.	3242
☐	L11	MCP-1RA OR MCP-1RB OR CCR2 OR CKR2 OR MCP-1 receptor	843
☐	L10	Coughlin.IN.	773
☐	L9	Coughlin-S.IN.	773
☐	L8	Coughlin-Shaun.IN.	1
☐	L7	Coughlin-S-R.IN.	11
☐	L6	Coughlin-Shaun-R.IN.	26
☐	L5	Charo-I-R.IN.	1
☐	L4	Charo-Israel-R.IN.	3
☐	L3	Charo.IN.	67
☐	L2	Charo-I.IN.	1
☐	L1	(Charo-Israel.IN.)	1

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☐	L4	= 1995	8
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☐	L2	Propagermanium OR eotaxin OR eotaxin-2 OR eotaxin-3 OR Amlodipine	2926
☐	L1	(rheumatoid arthritis OR alvoclitis OR artherosclerosis)	46717

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Search Results - Record(s) 1 through 8 of 8 returned.

☐ 1. Document ID: US 6806061 B1

Using default format because multiple data bases are involved.

L4: Entry 1 of 8 File: USPT Oct 19, 2004

US-PAT-NO: 6806061
DOCUMENT-IDENTIFIER: US 6806061 B1

TITLE: G protein-coupled receptor gene and methods of use therefor

DATE-ISSUED: October 19, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gerard; Craig J.	Dover	MA		
Gerard; Norma P.	Dover	MA		
Mackay; Charles R.	Newton Highlands	MA		
Ponath; Paul D.	Boston	MA		
Post; Theodore W.	Newton	MA		
Qin; Shixin	Lexington	MA		

US-CL-CURRENT: 435/69.1; 435/320.1, 435/325, 435/69.7, 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw Des

☐ 2. Document ID: US 6413967 B1

L4: Entry 2 of 8 File: USPT Jul 2, 2002

US-PAT-NO: 6413967
DOCUMENT-IDENTIFIER: US 6413967 B1
** See image for Certificate of Correction **

TITLE: Inhibition of novel calcium entry pathway in electrically non-excitable cells acting as an anti-proliferative therapy

DATE-ISSUED: July 2, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gray; Lloyd S.	Charlottesville	VA		
Haverstick; Doris M.	Charlottesville	VA		
Densmore; John J.	Charlottesville	VA		
Szabo; Gabor	Charlottesville	VA		

h e b b g e e e f e e ef b e

US-CL-CURRENT: 514/252.1; 514/252.12

ABSTRACT:

The present invention provides methods for screening for voltage gated (VG)-selective inhibitors, novel VG-selective inhibitors, compositions containing the same, methods for inhibiting calcium entry into electrically non-excitable cells with said VG-selective inhibitors, methods for preventing proliferation of electrically non-excitable cells with said VG-selective inhibitors as well as methods of treating autoimmune diseases, preventing graft rejections, preventing apoptosis and treating cancer with the same.

8 Claims, 17 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

☐ 3. Document ID: US 6265184 B1

L4: Entry 3 of 8 File: USPT Jul 24, 2001

US-PAT-NO: 6265184
DOCUMENT-IDENTIFIER: US 6265184 B1

TITLE: Polynucleotides encoding chemokine receptor 88C

DATE-ISSUED: July 24, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gray; Patrick W.	Seattle	WA		
Schweickart; Vicki L.	Seattle	WA		
Raport; Carol J.	Bothell	WA		

US-CL-CURRENT: 435/69.1; 435/252.3, 435/320.1, 435/325, 435/471, 435/71.1, 435/71.2, 530/350, 536/23.1, 536/23.5

ABSTRACT:

The present invention provides polynucleotides that encode the chemokine receptors 88-2B or 88C and materials and methods for the recombinant production of these two chemokine receptors. Also provided are assays utilizing the polynucleotides which facilitate the identification of ligands and modulators of the chemokine receptors. Receptor fragments, ligands, modulators, and antibodies are useful in the detection and treatment of disease states associated with the chemokine receptors such as atherosclerosis, rheumatoid arthritis, tumor growth suppression, asthma, and other inflammatory conditions.

11 Claims, 0 Drawing figures
Exemplary Claim Number: 1

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☐ 4. Document ID: US 5981230 A

L4: Entry 4 of 8 File: USPT Nov 9, 1999

US-PAT-NO: 5981230
DOCUMENT-IDENTIFIER: US 5981230 A

TITLE: Polynucleotide encoding chemokine .beta.-4

DATE-ISSUED: November 9, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Li; Haodong	Gaithersburg	MD		
Adams; Mark D.	North Potomac	MD		

US-CL-CURRENT: 435/69.5; 435/252.3, 435/320.1, 435/325, 435/471, 435/71.2, 530/324, 536/23.1, 536/23.5, 536/24.3, 536/24.31

ABSTRACT:

Human chemokine polypeptides and DNA (RNA) encoding such chemokine polypeptides and a procedure for producing such polypeptides by recombinant techniques is disclosed. Also disclosed are methods for utilizing such chemokine polypeptides for the treatment of leukemia, tumors, chronic infections, autoimmune disease, fibrotic disorders, wound healing and psoriasis. Antagonists against such chemokine polypeptides and their use as a therapeutic to treat rheumatoid arthritis, autoimmune and chronic inflammatory and infective diseases, allergic reactions, prostaglandin-independent fever and bone marrow failure are also disclosed. Diagnostic assays for identifying mutations in nucleic acid sequence encoding a polypeptide of the present invention and for detecting altered levels of the polypeptide of the present invention are also disclosed.

25 Claims, 4 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 3

☐ 5. Document ID: US 5935568 A

L4: Entry 5 of 8 File: USPT Aug 10, 1999

US-PAT-NO: 5935568
DOCUMENT-IDENTIFIER: US 5935568 A

TITLE: Gene therapy for effector cell regulation

DATE-ISSUED: August 10, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Dow; Steve W.	Denver	CO		
Elmslie; Robyn E.	Denver	CO		
Potter; Terence A.	Denver	CO		

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US-CL-CURRENT: 424/93.21; 435/375, 435/69.1, 514/44

ABSTRACT:

The present invention provides a nucleic acid-based therapeutic composition to treat an animal with disease by controlling the activity of effector cells, including T cells, macrophages, monocytes and/or natural killer cells, in the animal. Therapeutic compositions of the present invention include superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules, depending upon the disease being treated. The present invention also relates to an adjuvant for use with nucleic acid-based vaccines. Adjuvant compositions of the present invention include an immunogen combined with superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules.

28 Claims, 14 Drawing figures
Exemplary Claim Number: 1,3,5
Number of Drawing Sheets: 14

☐ 6. Document ID: US 5866373 A

L4: Entry 6 of 8 File: USPT Feb 2, 1999

US-PAT-NO: 5866373
DOCUMENT-IDENTIFIER: US 5866373 A

TITLE: Polynucleotide encoding a human chemotactic protein

DATE-ISSUED: February 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Li; Haodong	Gaithersburg	MD		
Ruben; Steven M.	Olney	MD		
Sutton, III; Granger G.	Columbia	MD		

US-CL-CURRENT: 435/69.5; 435/252.3, 435/254.11, 435/320.1, 435/325, 435/69.1, 435/91.41, 536/23.1, 536/23.5

ABSTRACT:

A human chemotactic protein polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide for preventing and/or treating for stem cell mobilization, myeloprotection and neuronal protection, to treat tumors, to promote wound healing, to combat parasitic infection and to regulate hematopoiesis. Also disclosed are antagonists against such polypeptides which may be employed to treat rheumatoid arthritis, lung inflammation, allergy, infectious diseases and to prevent inflammation and atherosclerosis. Diagnostic assays for identifying mutations in nucleic acid sequence encoding a polypeptide of the present invention and for detecting altered levels of the polypeptide of the present invention for detecting diseases are also disclosed.

25 Claims, 10 Drawing figures

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Exemplary Claim Number: 1
Number of Drawing Sheets: 8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc

☐ 7. Document ID: US 5668117 A

L4: Entry 7 of 8 File: USPT Sep 16, 1997

US-PAT-NO: 5668117
DOCUMENT-IDENTIFIER: US 5668117 A

TITLE: Methods of treating neurological diseases and etiologically related symptomology using carbonyl trapping agents in combination with previously known medicaments

DATE-ISSUED: September 16, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Shapiro; Howard K.	Narberth	PA	19072	

US-CL-CURRENT: 514/55; 436/518, 436/74, 514/1, 514/23, 514/54, 514/811, 514/866, 514/878, 514/879, 514/903, 514/912, 536/1.11, 536/20

ABSTRACT:

Therapeutic compositions comprising an effective amount of at least one carbonyl trapping agent alone or in combination with a therapeutically effective of a co-agent or medicament are disclosed. The compositions are used to treat a mammal suffering from a neurological disease characterized by covalent bond crosslinking between the nerve cells, other cellular structures and their intracellular and extracellular components, with disease induced carbonyl-containing aliphatic or aromatic hydrocarbons present in mammals.

29 Claims, 0 Drawing figures
Exemplary Claim Number: 1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc

☐ 8. Document ID: US 5478848 A

L4: Entry 8 of 8 File: USPT Dec 26, 1995

US-PAT-NO: 5478848
DOCUMENT-IDENTIFIER: US 5478848 A

TITLE: Inhibition of arthritis by L-type calcium channel antagonists nimodipine, nisoldipine and nifedipine

DATE-ISSUED: December 26, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY

h e b b g e e e f e e ef b e

Aune; Thomas M. Hamden CT

US-CL-CURRENT: 514/356; 514/355

ABSTRACT:

The present invention comprises new methods for treating rheumatoid arthritis. It has been found that the L-type calcium channel antagonists are effective in treating arthritis. Nimodipine, nisoldipine, and nifedipine, are examples of specific compounds useful in the present invention.

8 Claims, 1 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 1

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The amino-terminal domain of the CCR2 chemokine receptor acts as coreceptor for HIV-1 infection.
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Receptors for IgG complexes activate synthesis of monocyte

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1: Lab Invest. 1994 May;70(5):631-8.

Cytokine expression, upregulation of intercellular adhesion molecule-1, and leukocyte infiltration in experimental tubulointerstitial nephritis.

Tang WW, Feng L, Mathison JC, Wilson CB.

Department of Immunology, Scripps Research Institute, La Jolla, California.

BACKGROUND: Cytokines are intercellular polypeptide messengers that mediate immune and inflammatory responses. The temporal profile of interleukin-1 beta (IL-1 beta), IL-6, tumor necrosis factor alpha (TNF-alpha), and monocyte chemotactic protein 1 (MCP-1) expression was examined in anti-tubular basement membrane (TBM) antibody-associated tubulointerstitial nephritis (TIN). EXPERIMENTAL DESIGN: TIN was induced by immunization of Brown Norway rats with bovine cortical TBM, whereas control rats received ovalbumin. Whole kidney RNA was assessed with the RNase protection assay 3, 7, 8, 9, 10, 12, and 14 days after immunization. Cytokine mRNA expression was correlated with TNF-alpha bioactivity, renal intercellular adhesion molecule-1 expression, and CD18-positive leukocyte infiltration by immunohistochemistry. RESULTS: Increased IL-1 beta, TNF-alpha, and MCP-1 mRNA relative to glyceraldehyde-3-phosphate dehydrogenase appeared on day 7 when TIN involved 10 to 40% of the cortex, and peaked rapidly on day 8 when there was 60 to 80% cortical involvement (at which time 75 to 80% of the infiltrating cells were neutrophils). The increase in TNF-alpha mRNA correlated with increased bioactivity. The influx of mononuclear cells on day 8 was preceded by the expression of MCP-1 mRNA. The infiltrating leukocytes expressed the leukocyte beta 2-integrin (CD18) and were found in areas with increased intercellular adhesion molecule-1 expression. The mRNAs for IL-1 beta, TNF-alpha, and MCP-1 were undetectable by day 10 (at which time 95% of the infiltrating cells were mononuclear). An increase in IL-1 receptor antagonist mRNA paralleled those of IL-1 beta. The expression of IL-6 mRNA was similar to that for IL-1, except that it disappeared by day 9. CONCLUSIONS: There is a temporal association in the expression of IL-1 beta, TNF alpha, MCP-1, and IL-6 with the upregulation of intercellular adhesion molecule-1 and leukocyte infiltration within the tubulointerstitium in anti-TBM antibody-associated TIN. The narrow window of time through which these cytokines are expressed and the coincidence of their peak expression on day 8 suggest complex cytokine interactions in the pathogenesis of anti-TBM antibody TIN.

1: J Biol Chem. 1994 Jul 1;269(26):17730-3.

Identification and characterization of a promiscuous chemokine-binding protein in a human erythroleukemic cell line.

Horuk R, Wang ZX, Peiper SC, Hesselgesser J.

Department of Protein Chemistry, Genentech Inc., South San Francisco, California 94080.

The erythrocyte chemokine receptor is a cell surface protein that binds a wide array of chemokines including interleukin-8 (IL-8), melanoma growth stimulating activity (MGSA), monocyte chemotactic protein-1 (MCP-1), and RANTES (Regulated on Activation, Normal T Expressed and Secreted). This protein has also been identified as the Duffy blood group antigen, a cell surface receptor for the malarial parasite Plasmodium vivax. In the present study, we have identified a chemokine receptor-like binding protein in a human erythroleukemic cell line (HEL), which, based on its molecular properties, may be related to the erythrocyte chemokine receptor. Saturation binding studies with 125I-IL-8 revealed a single class of IL-8 binding sites in HEL cells with a KD of 7.4 +/- 1.9 nM and a receptor density of 12,818 +/- 965 binding sites/cell. In competition studies unlabeled IL-8 MGSA, MCP-1, and RANTES were fully able to inhibit the binding of 125I-IL-8 to HEL cells. Chemical cross-linking with radiolabeled IL-8 resulted in a cross-linked species of 60 kDa in membranes from HEL cells. The labeling was specific since it was inhibited by pre-incubation with 1 microM unlabeled IL-8 or MGSA. A monoclonal antibody (Fy6) to the human erythrocyte Duffy blood group antigen/chemokine receptor blocked the binding of IL-8 and other chemokines to the HEL cell chemokine receptor-like binding protein. Cell membranes from HEL cells and from erythrocyte ghosts were subjected to SDS-PAGE and analyzed by Western blotting with anti-Fy6. The antibody bound to a molecule with a molecular mass of 50 kDa in HEL cell membranes and 40 kDa in erythrocyte ghosts. Northern blot analysis of mRNA revealed that the HEL chemokine-binding protein hybridized to a cDNA probe to the Duffy antigen/chemokine receptor.

PMID: 7517400 [PubMed - indexed for MEDLINE]

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1: Kidney Int. 1996 Mar;49(3):715-21.

Differential expression of macrophage inflammatory protein-2 and monocyte chemoattractant protein-1 in experimental glomerulonephritis.

Tam FW, Karkar AM, Smith J, Yoshimura T, Steinkasserer A, Kurrle R, Langner K, Rees AJ.

Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London, England, United Kingdom.

We examined the relation between glomerular expression of chemokines from alpha-subfamily (macrophage inflammatory protein-2, MIP-2) and beta-subfamily (monocyte chemoattractant protein-1, MCP-1) and infiltration of neutrophils and monocytes in antibody mediated glomerulonephritis in rats. In the accelerated model of nephrotoxic nephritis (NTN), glomerular expression of MIP-2 and MCP-1 genes correlated with the sequential migration of neutrophil and monocyte influx, respectively. These relationships were investigated further in the heterologous phase of NTN by applying various treatments known to modulate the severity of injury. Pretreatment with bacterial lipopolysaccharide resulted in greater injury, MIP-2 expression increased 25- to 50-fold, and the glomerular neutrophil count increased two- to fourfold. Both MIP-2 mRNA levels and neutrophil infiltration were reduced by additional pretreatment with IL-6, IL-1 receptor antagonist, soluble IL-1 receptor or soluble TNF receptor (Spearman correlation coefficient $r = 0.897$, $P < 0.005$). In the heterologous phase of NTN, different pre-treatments only resulted in trivial changes in MCP-1 expression and monocyte infiltration. In conclusion, glomerular MIP-2 gene expression correlates with neutrophil infiltration both temporally during the evolution of nephritis, and when glomerular injury is modified by treatment. Glomerular MCP-1 gene expression correlates with monocyte influx. The data show chemokines of alpha- and beta-subfamilies co-operative to cause selective and sequential migration of different leukocyte subsets during development of antibody mediated glomerulonephritis.

PMID: 8648912 [PubMed - indexed for MEDLINE]

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1: J Gastroenterol. 1996 Dec;31(6):907-16.

Alterations of the mucosal immune system in inflammatory bowel disease.

MacDermott RP.

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The normal intestinal immune system is under a balance in which proinflammatory and anti-inflammatory cells and molecules are carefully regulated to promote a normal host mucosal defense capability without destruction of intestinal tissue. Once this careful regulatory balance is disturbed, nonspecific stimulation and activation can lead to increased amounts of potent destructive immunologica and inflammatory molecules being produced and released. The concept of balance and regulation of normal mucosal immune and inflammatory events is indicative of how close the intestine is to developing severe inflammation. The normal intestinal mucosal immune system is constantly stimulated by lumenal contents and bacteria. The stimulatory molecules present in the intestinal lumen that activate and induce subsequent mucosal immunologic and inflammatory events include bacterial cell wall products, such as peptidoglycans and lipopolysaccharides, as well as other chemotactic and toxic bacterial products that are produced by the many different types of bacteria within the gastrointestinal tract. These highly stimulatory bacterial cell wall products are capable of activating macrophages and T lymphocytes to release potent proinflammatory cytokines, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-alpha). IL-1, IL-6, and TNF-alpha increase the presence of human leukocyte antigen (HLA) class II antigen-presenting molecules on the surfaces of epithelial cells, endothelial cells, macrophages, and B cells, thus increasing their ability to present lumenal antigens and bacterial products. The proinflammatory cytokines IL-1 and TNF-alpha also increase the ability of epithelial cells, endothelial cells, macrophages, and fibroblasts to secrete potent chemotactic cytokines, such as interleukin-8 (IL-8) and monocyte chemoattractant protein-1 (MCP-1), which serve to increase the movement of macrophages and granulocytes from the circulation into the inflamed mucosa. Thus, through lumenal exposure to potent, nonspecific stimulatory bacterial products, the state of activation of the intestinal immune system and mucosal inflammatory pathways are markedly up-regulated. This raises the question of whether there is a deficiency in effective down-regulation through the absence of normally suppressive cytokines such as interleukin-10 (IL-10),

transforming growth factor-beta (TGF-beta), interleukin-4 (IL-4), and IL-1 receptor antagonist. Normally, the turning off of the active and destructive immunologic and inflammatory events should occur following the resolution of a bacterial or viral infection that has been appropriately defended against and controlled by the mucosal immune system. In inflammatory bowel disease (IBD), however, the down-regulatory events and processes that should turn off the immunologic and inflammatory protective processes, once the pathogenic agent has been cleared, appear to be deficient or only partially effective. We may find that we ultimately are dealing with disease processes that have more than one genetic or cellular basis. The improved understanding of the immunopathophysiology of IBD will allow exploration of novel immunologic and genetic approaches, such as gene replacement therapy, administration of a suppressor cytokine or an altered cell surface antigen, the administration of humanized monoclonal antibodies directed against proinflammatory cytokines, or the development of newer strategies against fundamental cell biologic mechanisms such as adhesion molecules.

Publication Types:

- Review
- Review, Tutorial

PMID: 9027661 [PubMed - indexed for MEDLINE]

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1: J Exp Med. 1998 Jul 6;188(1):157-67.

The coordinated action of CC chemokines in the lung orchestrates allergic inflammation and airway hyperresponsiveness.

Gonzalo JA, Lloyd CM, Wen D, Albar JP, Wells TN, Proudfoot A, Martinez-A C, Dorf M, Bjerke T, Coyle AJ, Gutierrez-Ramos JC.

Millennium Pharmaceuticals, Inc., Cambridge, Massachusetts 02139, USA.

The complex pathophysiology of lung allergic inflammation and bronchial hyperresponsiveness (BHR) that characterize asthma is achieved by the regulated accumulation and activation of different leukocyte subsets in the lung. The development and maintenance of these processes correlate with the coordinated production of chemokines. Here, we have assessed the role that different chemokines play in lung allergic inflammation and BHR by blocking their activities in vivo. Our results show that blockage of each one of these chemokines reduces both lung leukocyte infiltration and BHR in a substantially different way. Thus, eotaxin neutralization reduces specifically BHR and lung eosinophilia transiently after each antigen exposure. Monocyte chemoattractant protein (MCP)-5 neutralization abolishes BHR not by affecting the accumulation of inflammatory leukocytes in the airways, but rather by altering the trafficking of the eosinophils and other leukocytes through the lung interstitium. Neutralization of RANTES (regulated upon activation, normal T cell expressed and secreted) receptor(s) with a receptor antagonist decreases significantly lymphocyte and eosinophil infiltration as well as mRNA expression of eotaxin and RANTES. In contrast, neutralization of one of the ligands for RANTES receptors, macrophage-inflammatory protein 1alpha, reduces only slightly lung eosinophilia and BHR. Finally, MCP-1 neutralization diminishes drastically BHR and inflammation, and this correlates with a pronounced decrease in monocyte- and lymphocyte-derived inflammatory mediators. These results suggest that different chemokines activate different cellular and molecular pathways that in a coordinated fashion contribute to the complex pathophysiology of asthma, and that their individual blockage results in intervention at different levels of these processes.

PMID: 9653092 [PubMed - indexed for MEDLINE]

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1: Rheumatology (Oxford). 2004 Sep;43(9):1121-8. Epub 2004 Jun 22.

Anti-monocyte chemoattractant protein-1 gene therapy attenuates nephritis in MRL/lpr mice.

Shimizu S, Nakashima H, Masutani K, Inoue Y, Miyake K, Akahoshi M, Tanaka Y, Egashira K, Hirakata H, Otsuka T, Harada M.

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OBJECTIVE: Monocyte chemoattractant protein-1 (MCP-1) is up-regulated and recruits and activates inflammatory cells in human diffuse proliferative lupus nephritis (DPLN) and in nephritis of lupus model MRL/lpr mice. The aim of this study was to examine whether anti-MCP-1 gene therapy inhibits the progression of nephritis in MRL/lpr mice. METHOD: An NH(2)-terminal deletion mutant of the MCP-1 gene, 7ND, was injected into skeletal muscles of MRL/lpr mice with advanced stage nephritis to blockade MCP-1 and its receptor (CCR2) signalling pathway. RESULT: Histological findings of kidneys in treated mice, which received more than four injections of 7ND, showed that protection against renal injury resulted from reduced infiltration of leucocytes. Therefore, this therapy has been shown to prolong the life span of MRL/lpr mice. CONCLUSION: Anti-MCP-1 gene therapy is specifically effective in the localized inflammatory region. The data presented here indicate that this anti-MCP-1 gene therapy may be effective adjunct in the management of DPLN.

PMID: 15213333 [PubMed - indexed for MEDLINE]

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1: Ann Med. 2004;36(2):98-118.

Chemokines and atherosclerosis.

Sheikine Y, Hansson GK.

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Atherosclerosis is an inflammatory disease of the vessel wall, characterized by the accumulation of leukocytes, especially macrophages and T-cells. Chemokines are small heparin-binding polypeptides, whose main function is to attract cells to the areas of developing inflammation. They function by ligating G-protein coupled chemokine receptors initiating different signaling cascades. In vivo and in vitro investigations showed that chemokines are produced by a variety of cells and play important roles in the development and progression of many physiological and pathological conditions including atherosclerosis. Chemokines such as MCP-1, MCP-4, MIP-1 and RANTES may mediate leukocyte trafficking to, and their retention in, the plaque while CXCL16 seems to fulfill the dual function of a chemokine and a scavenger receptor. Chemokine and chemokine receptor homologues are secreted by several viruses, which may also play a role in the pathogenesis of atherosclerosis. Expression levels and gene polymorphisms of some chemokines may become useful clinical markers of atherosclerosis and other cardiovascular diseases. Modulation of chemokines and chemokine receptors' expression as well as their signaling pathways may provide important anti-atherogenic strategies.

Publication Types:
- Review
- Review, Tutorial

PMID: 15119830 [PubMed - indexed for MEDLINE]

1: J Lab Clin Med. 2003 Sep;142(3):187-95.

Experimental hypersensitivity pneumonitis: role of MCP-1.

Schuyler M, Gott K, Cherne A.

Department of Medicine, Albequerque Veterans Affairs Medical Center, University of New Mexico, 87108, USA.

Inhalation of Saccharopolyspora rectivirgula causes "farmer's lung" disease, a classic example of hypersensitivity pneumonitis (HP). Monocyte chemoattractant protein-1 (MCP-1) is increased in the bronchoalveolar lavage fluid of mice challenged with S rectivirgula, and S rectivirgula induces MCP-1 secretion by alveolar macrophages. We tested the hypothesis that MCP-1 and its receptor CC chemokine receptor-2 (CCR2) are essential to the development of experimental HP by treating mice with MCP-1 antibody and using CCR2(-/-) mice. Administration of anti-MCP-1 did not change the response to intratracheally administered S rectivirgula. CCR2(-/-) animals responded in a fashion similar to that of wild-type animals to intratracheally administered.S rectivirgula. To determine the influence of the MCP-1-CCR2 interaction in vitro, we transferred S rectivirgula-cultured spleen cells from S rectivirgula-sensitized mice, to naive recipients. Later, challenge of the recipients with intratracheal S rectivirgula and examination of both lung histology and bronchoalveolar lavage fluid characteristics were used to determine whether adoptive transfer had occurred. We found that cultured cells from CCR2(-/-) animals were fully capable of adoptive transfer. We conclude that interaction of MCP-1 with CCR2 is not necessary for the development of pulmonary inflammation in response to intratracheally administered S rectivirgula or cells able to adoptively transfer experimental HP.

PMID: 14532907 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg c e e e b b e b c

1: Circ Res. 2002 Mar 8;90(4):488-94.

Targeting CCR2 or CD18 inhibits experimental in-stent restenosis in primates: inhibitory potential depends on type of injury and leukocytes targeted.

Horvath C, Welt FG, Nedelman M, Rao P, Rogers C.

Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA.

A central role for leukocytes in neointimal hyperplasia after arterial injury is suspected. However, the relative importance of neutrophils and monocytes in balloon or stent-induced injury are not well understood, and mechanistic targeting of leukocyte recruitment or function is crude. We determined the temporal and spatial distribution of different leukocytes after balloon and stent-induced injury in primate iliac arteries. Based on these data, we targeted neutrophil and monocyte recruitment selectively after angioplasty or stent implantation and demonstrated that monocyte-specific blockade achieved via blockade of the MCP-1 receptor CCR2, was effective at reducing neointimal hyperplasia after stenting. In contrast, combined neutrophil and monocyte blockade achieved by targeting the leukocyte beta(2)-integrin beta-subunit CD18 was required to reduce neointimal hyperplasia after balloon injury. Distinct patterns of leukocyte infiltration in balloon versus stent-injured arteries predict distinct mechanisms for antiinflammatory strategies targeting neutrophils or monocytes in primates and may assist design of effective clinical strategies for optimizing vascular interventions.

PMID: 11884380 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg c e e e b b e b c

1: J Interferon Cytokine Res. 2001 Jun;21(6):389-98.

An anti-inflammatory drug, propagermanium, may target GPI-anchored proteins associated with an MCP-1 receptor, CCR2.

Yokochi S, Hashimoto H, Ishiwata Y, Shimokawa H, Haino M, Terashima Y, Matsushima K.

Central Research Laboratory, Sanwa Kagaku Kenkyusho Co., Ltd., Hokusei-cho, Inabe-gun, Mie 511-0406, Japan.

Monocyte chemoattractant protein-1 (MCP-1) promotes the migration and activation of monocytes and plays a pivotal role in the development of chronic inflammation. Propagermanium (3-oxygermylpropionic acid polymer) has been used as a therapeutic agent against chronic hepatitis B in Japan. We report here that propagermanium specifically inhibits in vitro chemotactic migration of monocytes by MCP-1. Propagermanium did not inhibit binding of MCP-1 to a human monocytic cell line, THP-1 cells, or affect intracellular Ca(2+) mobilization or the cAMP concentration in MCP-1-treated THP-1 cells. The effect of propagermanium seems to require glycosylphosphatidylinositol (GPI)-anchored proteins, as cleavage of GPI anchors by phosphatidylinositol-phospholipase C (PI-PLC) eliminated the inhibitory activity of propagermanium. Anti-GPI-anchored protein antibodies, such as anti-CD55 and anti-CD59, reduced staining of C-C chemokine receptor 2 (CCR2) with an anti-CCR2 antibody against the N-terminus of CCR2 in a flow cytometric analysis, and these antibodies also selectively inhibited MCP-1-induced migration of THP-1 cells. Furthermore, under fluorescence microscopy, GPI-anchored proteins colocalized with CCR2 on THP-1 cells. These results suggest that propagermanium may target GPI-anchored proteins that are closely associated with CCR2 to selectively inhibit the MCP-1-induced chemotaxis, thus providing a mechanistic basis for the anti-inflammatory effects of the drug.

PMID: 11440636 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg c e e e b b e b c

1: J Biochem (Tokyo). 2001 Apr;129(4):577-83. Related Articles, Links

Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

In this study, we attempted to analyze the peptide motifs recognized by 24822.111 and F9, monoclonal antibodies (mAbs) that inhibit the chemotactic activity of monocyte chemoattractant protein-1 (MCP-1), a member of the CC subfamily of chemokines. We isolated phage clones from a phage display library and identified six peptide motifs. One of these clones, C27, was strongly and specifically recognized by 24822.111 mAb, while another, G25, was similarly recognized by F9 mAb. Both the C27 motif and the G25 motif contain two cysteines in their sequences and have little homology to the primary amino acid sequence of MCP-1. These clones, however, bound to THP-1 cells, and the binding was competitively inhibited by MCP-1. The clones strongly inhibited the MCP-1-induced chemotaxis of human monocytes. The synthetic and intramolecularly disulfide-linked peptides of C27 and G25 (sC27 and sG25) also inhibited the chemotaxis induced by MCP-1, while their derivatives with serine in place of cysteine did not, suggesting the importance of the loop structure for the inhibition. These results suggest that sC27 and sG25 may mimic the MCP-1-binding domain to the MCP-1 receptor.

PMID: 11275557 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: J Biochem (Tokyo). 2001 Apr;129(4):577-83. Related Articles, Links

Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

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PMID: 11275557 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: J Neuroimmunol. 2000 Aug 1;108(1-2):192-200.

Expression of the beta-chemokine receptors CCR2, CCR3 and CCR5 in multiple sclerosis central nervous system tissue.

Simpson J, Rezaie P, Newcombe J, Cuzner ML, Male D, Woodroofe MN.

Biomedical Research Centre and Division of Biomedical Sciences, Sheffield Hallam University, City Campus, Pond Street, South Yorkshire, S1 1WB, Sheffield, UK.

Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system (CNS) characterised by perivascular inflammatory cell infiltrates and plaques of demyelination. Chemokines have been shown to play an important role in the activation and directional migration of cells to sites of CNS inflammation. The action of chemokines requires the expression of their complementary chemokine receptors by their target cells. We have examined the expression of the beta-chemokine receptors CCR2, CCR3 and CCR5 in post-mortem MS CNS tissue using single- and double-labelling immunocytochemistry techniques. Low levels of CCR2, CCR3 and CCR5 were expressed by microglial cells throughout control CNS tissue. In chronic active MS lesions CCR2, CCR3 and CCR5 were associated with foamy macrophages and activated microglia. CCR2 and CCR5 were also present on large numbers of infiltrating lymphocytes. A smaller number of CCR3-positive lymphocytes were present, but we also noted CCR3 and CCR5 on astrocytes in five of the 14 cases of MS investigated, particularly associated with processes around vessels and at the glia limitans. Ligands for CCR2 and CCR3 include MCP-1 and MCP-3 which were co-localised around vessels with the infiltrating leukocytes, but were also present in unaffected areas of cortex. The elevated expression of CCR2, CCR3 and CCR5 in the CNS in MS suggests these beta-chemokine receptors and their ligands play a role in the pathogenesis of MS.

PMID: 10900353 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg c e e e b b e b c

1: Blood. 2000 Jul 1;96(1):34-40.

Human endothelial cells express CCR2 and respond to MCP-1: direct role of MCP-1 in angiogenesis and tumor progression.

Salcedo R, Ponce ML, Young HA, Wasserman K, Ward JM, Kleinman HK, Oppenheim JJ, Murphy WJ.

Laboratory of Molecular Immunoregulation, Laboratory of Experimental Immunology, Division of Basic Sciences; Intramural Research Support Program, SAIC, Frederick, MD, USA.

Although several CXC chemokines have been shown to induce angiogenesis and play roles in tumor growth, to date, no member of the CC chemokine family has been reported to play a direct role in angiogenesis. Here we report that the CC chemokine, monocyte chemotactic protein 1 (MCP-1), induced chemotaxis of human endothelial cells at nanomolar concentrations. This chemotactic response was inhibited by a monoclonal antibody to MCP-1. MCP-1 also induced the formation of blood vessels in vivo as assessed by the chick chorioallantoic membrane and the matrigel plug assays. As expected, the angiogenic response induced by MCP-1 was accompanied by an inflammatory response. With the use of a rat aortic sprouting assay in the absence of leukocytic infiltrates, we ruled out the possibility that the angiogenic effect of MCP-1 depended on leukocyte products. Moreover, the direct effect of MCP-1 on angiogenesis was consistent with the expression of CCR2, the receptor for MCP-1, on endothelial cells. Assessment of supernatant from a human breast carcinoma cell line demonstrated the production of MCP-1. Treatment of immunodeficient mice bearing human breast carcinoma cells with a neutralizing antibody to MCP-1 resulted in significant increases in survival and inhibition of the growth of lung micrometastases. Taken together, our data indicate that MCP-1 can act as a direct mediator of angiogenesis. As a chemokine that is abundantly produced by some tumors, it can also directly contribute to tumor progression. Therefore, therapy employing antagonists of MCP-1 in combination with other inhibitors of angiogenesis may achieve more comprehensive inhibition of tumor growth.

PMID: 10891427 [PubMed - indexed for MEDLINE]

1: Am J Pathol. 2000 Apr;156(4):1245-52.

Exaggerated hepatic injury due to acetaminophen challenge in mice lacking C-C chemokine receptor 2.

Hogaboam CM, Bone-Larson CL, Steinhauser ML, Matsukawa A, Gosling J, Boring L, Charo IF, Simpson KJ, Lukacs NW, Kunkel SL.

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Monocyte chemoattractant protein-1 is one of the major C-C chemokines that has been implicated in liver injury. The C-C chemokine receptor, CCR2, has been identified as the primary receptor that mediates monocyte chemoattractant protein-1 (MCP-1) responses in the mouse. Accordingly, the present study addressed the role of CCR2 in mice acutely challenged with acetaminophen (APAP). Mice genetically deficient in CCR2 (CCR2(-/-)) and their wild-type counterparts (CCR2(+/+)) were fasted for 10 hours before receiving an intraperitoneal injection of APAP (300 mg/kg). Liver and serum samples were removed from both groups of mice before and at 24 and 48 hours post APAP. Significantly elevated levels of MCP-1 were detected in liver samples from CCR2(+/+) and CCR2(-/-) mice at 24 hours post-APAP. Although CCR2(+/+) mice exhibited no liver injury at any time after receiving APAP, CCR2(-/-) mice exhibited marked evidence of necrotic and TUNEL-positive cells in the liver, particularly at 24 hours post-APAP. Enzyme-linked immunosorbent assay analysis of liver homogenates from both groups of mice at the 24 hours time point revealed that liver tissue from CCR2(-/-) mice contained significantly greater amounts of immunoreactive IFN-gamma and TNF-alpha. The in vivo immunoneutralization of IFN-gamma or TNF-alpha significantly attenuated APAP-induced liver injury in CCR2(-/-) mice and increased hepatic IL-13 levels. Taken together, these findings demonstrate that CCR2 expression in the liver provides a hepatoprotective effect through its regulation of cytokine generation during APAP challenge.

PMID: 10751350 [PubMed - indexed for MEDLINE]

1: Mol Immunol. 1999 Dec;36(18):1249-54.

Inhibition of M-tropic HIV-1 infection by the fd phage-gene 3 protein with MIP-1alpha-binding activity.

Meta A, Torigoe N, Ito Y, Arakaki R, Nakashima H, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, 1-21-40 Korimoto, Kagoshima, Japan.

CCR5 is a chemokine receptor with seven transmembrane-domains. It is expressed on T cells and macrophages and functions as the principal co-receptor for macrophage (M)-tropic strains of HIV-1. The anti-CCR5 monoclonal antibody (mAb) 2D7 inhibits the binding and chemotaxis of the three natural beta-chemokine ligands of CCR5, macrophage inflammatory protein (MIP)-1alpha, MIP-1beta, and RANTES, to CCR5(+) cells. The mAb also efficiently blocks the infectivity of several M-tropic and dual-tropic HIV-1 strains in vitro.In this study, we attempted to determine the peptide motif recognized with the 2D7 mAb. We isolated phage clones by panning a phage display library using 2D7 and identified three peptide motifs. One of these phage clones (M23) showed a marked inhibitory activity on HIV-1 infection. The unique sequence of 15 amino acids with an internal disulfide bond was inserted in the g3p of the M23 phage clone (M23-g3p). The M23-g3p was purified by fast-performance liquid chromatography (FPLC). We show here that (1) M23-g3p was specifically recognized with anti-CCR5 mAb; (2) M23-g3p showed inhibitory activity on the infectivity of M-tropic but not T-tropic HIV-1 strains; (3) M23-g3p bound to MIP-1alpha, MIP-1beta, and RANTES but not MCP-1. These results suggested that the M23-g3p might mimic the CCR5-binding domain shared by beta-chemokines, MIP-1alpha, MIP-1beta, and RANTES as well as the HIV-1 infection.

PMID: 10684964 [PubMed - indexed for MEDLINE]

1: J Immunol. 1999 Oct 1;163(7):3969-75.

Intracellular IL-1 receptor antagonist is elevated in human dermal fibroblasts that overexpress intracellular precursor IL-1 alpha.

Higgins GC, Wu Y, Postlethwaite AE.

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Cultured dermal fibroblasts from systemic sclerosis patients express higher levels of intracellular IL-1 alpha than fibroblasts from healthy controls. In this study, we found that systemic sclerosis dermal fibroblasts also express higher levels of the intracellular isoform of IL-1 receptor antagonist (icIL-1Ra) than normal fibroblasts after stimulation with IL-1 beta or TNF-alpha. A possible relationship between elevated precursor IL-1 alpha (preIL-1 alpha) and elevated icIL-1Ra was investigated by transducing normal dermal fibroblasts to overexpress preIL-1 alpha, preIL-1 beta, or icIL-1Ra. Fibroblasts that overexpressed icIL-1Ra did not have elevated levels of IL-1 alpha. On the other hand, fibroblasts that overexpressed preIL-1 alpha had at least 4-fold higher basal levels of icIL-1Ra than control fibroblasts and 4-fold higher levels of icIL-1Ra after induction with IL-1 beta or TNF-alpha. Fibroblasts overexpressing preIL-1 beta did not exhibit elevated icIL-1Ra. The differences in icIL-1Ra protein levels were reflected in differences in mRNA. In contrast, IL-1-stimulated levels of MCP-1 and IL-6 were not different in control and preIL-1 alpha-transduced fibroblasts. Addition of neutralizing anti-IL-1 alpha Abs to fibroblast cultures did not diminish basal or stimulated levels of icIL-1Ra in the preIL-1 alpha-transduced cells, supporting an intracellular site of action of preIL-1 alpha. This is the first report of an association between intracellular levels of these IL-1 family members. We hypothesize that intracellular preIL-1 alpha participates in the regulation of icIL-1Ra.

PMID: 10490999 [PubMed - indexed for MEDLINE]

All: 5

Items 1 - 5

One page.

1: Zee RY, Cook NR, Cheng S, Erlich HA, Lindpaintner K, Lee RT, Ridker PM. Related Articles, Links

Threonine for alanine substitution in the eotaxin (CCL11) gene and the risk of incident myocardial infarction.
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PMID: 15186951 [PubMed - indexed for MEDLINE]

2: Economou E, Tousoulis D, Katinioti A, Stefanadis C, Trikas A, Pitsavos C, Tentolouris C, Toutouza MG, Toutouzas P. Related Articles, Links

Chemokines in patients with ischaemic heart disease and the effect of coronary angioplasty.
Int J Cardiol. 2001 Aug;80(1):55-60.
PMID: 11532547 [PubMed - indexed for MEDLINE]

3: Ensminger SM, Spriewald BM, Sorensen HV, Witzke O, Flashman EG, Bushell A, Morris PJ, Rose ML, Rahemtulla A, Wood KJ. Related Articles, Links

Critical role for IL-4 in the development of transplant arteriosclerosis in the absence of CD40-CD154 costimulation.
J Immunol. 2001 Jul 1;167(1):532-41.
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4: Haley KJ, Lilly CM, Yang JH, Feng Y, Kennedy SP, Turi TG, Thompson JF, Sukhova GH, Libby P, Lee RT. Related Articles, Links

Overexpression of eotaxin and the CCR3 receptor in human atherosclerosis: using genomic technology to identify a potential novel pathway of vascular inflammation.
Circulation. 2000 Oct 31;102(18):2185-9.
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Cloning, in vitro expression, and functional characterization of a novel human CC chemokine of the monocyte chemotactic protein (MCP) family (MCP-4) that binds and signals through the CC chemokine receptor 2B.
J Biol Chem. 1997 Jun 27;272(26):16404-13.
PMID: 9195948 [PubMed - indexed for MEDLINE]

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Items 1 - 3

One page.

1: Shimokawa H, Eto Y, Miyata K, Morishige K, Kandabashi T, Matsushima K, Takeshita A. Related Articles, Links

Propagermanium suppresses macrophage-mediated formation of coronary arteriosclerotic lesions in pigs in vivo.
J Cardiovasc Pharmacol. 2003 Mar;41(3):372-80.
PMID: 12605015 [PubMed - indexed for MEDLINE]

2: Eto Y, Shimokawa H, Tanaka E, Morishige K, Fuchigami M, Ishiwata Y, Matsushima K, Takeshita A. Related Articles, Links

Long-term treatment with propagermanium suppresses atherosclerosis in WHHL rabbits.
J Cardiovasc Pharmacol. 2003 Feb;41(2):171-7.
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Propagermanium reduces atherosclerosis in apolipoprotein E knockout mice via inhibition of macrophage infiltration.
Arterioscler Thromb Vasc Biol. 2002 Jun 1;22(6):969-74.
PMID: 12067906 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg e ch b e

1: Am J Cardiol. 1989 Nov 7;64(17):21I-34I.

Amlodipine, a new 1,4-dihydropyridine calcium antagonist with a particularly strong antihypertensive profile.

Fleckenstein A, Frey M, Zorn J, Fleckenstein-Grun G.

Physiological Institute, University of Freiburg, Federal Republic of Germany.

The effects of a new 1,4-dihydropyridine derivative amlodipine have been compared with results from our previous work. Application of amlodipine at a concentration of 1.6 X 10(-6) M to isolated guinea-pig papillary muscle for 120 minutes produced a 50% reduction in tension development compared with a concentration of 3.7 X 10(-7) M nifedipine needed to produce the same result under identical conditions. This suggests that amlodipine has even weaker negative inotropic effects than nifedipine. In isolated porcine coronary strips, the K+-induced contractions were approximately 10,000 times more sensitive to the relaxing effects of nisoldipine, nitrendipine and nicardipine than to those of papaverine, whereas nifedipine and amlodipine were 3,000 times more potent than papaverine. However, in comparison with these in vitro actions, the efficacy of amlodipine appears to be greater in vivo: Simultaneous subcutaneous injection of nifedipine (20 mg/kg) and of equimolar doses of nisoldipine and felodipine attenuated the myocardial calcium uptake by rat hearts in situ (stimulated with a single subcutaneous dose of 30 mg/kg isoproterenol) with the same efficacy, whereas the actions of nitrendipine and nimodipine were considerably weaker. In contrast, amlodipine antagonized isoproterenol-stimulated myocardial calcium accumulation more effectively. Furthermore, amlodipine exhibited a high antihypertensive potency combined with rapid onset and long duration of action: Amlodipine (10 mg/kg orally [p.o.]) reduced the blood pressure of spontaneously hypertensive rats almost to the same extent as nifedipine, nitrendipine, verapamil and felodipine administered at the much higher doses of 100 mg/kg p.o. Amlodipine (20 mg/kg/day p.o.) maintained normal blood pressure during the whole life span of Dahl-S rats (5 months), but this dose is considerably lower than that reported for other 1,4-dihydropyridines. The survival of NaCl-loaded Dahl-S rats increased from 20 to 100% after administration of amlodipine (20 mg/kg/day p.o.) over 10 weeks: The effective dose of other calcium antagonists is approximately 5 times higher, but well tolerated as, e.g., demonstrated in long-term studies on Dahl-S rats with nitrendipine over 12 months. Increases in systemic arteriolar tone can be visualized in the ocular fundus of spontaneously hypertensive rats. After amlodipine (10 mg/kg p.o.) arteriolar spasm declines. Prophylaxis with 2

doses of 20 mg/kg amlodipine daily in NaCl-loaded Dahl-S rats abolished the macroscopic and histologic changes that are normally seen in branches of the mesenteric artery. With use of electron microscopy, calcium accumulation in the lamina elastica interna was demonstrated by the potassium-pyroantimonate technique.(ABSTRACT TRUNCATED AT 400 WORDS)

PMID: 2530884 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: Am J Cardiol. 1990 Nov 20;66(18):2H-9H.

Progress in cardioprotection: the role of calcium antagonists.

Kloner RA, Przyklenk K.

Heart Institute, Hospital of the Good Samaritan, Los Angeles, California 90017.

Calcium antagonists are now widely used for the treatment of clinical hypertension and angina pectoris. They are efficacious for the treatment of vasospastic, fixed atherosclerotic and mixed angina; they reduce the incidence of silent ischemia; and they have been shown to reduce postmyocardial infarct angina. Experimental data suggest that they may have certain cardioprotective properties in cases of acute myocardial ischemia and infarction, stunned myocardium, diastolic dysfunction, left ventricular hypertrophy and atherosclerosis. Moreover, they have been shown to improve exercise performance, as well as the diastolic abnormalities in patients with hypertrophic cardiomyopathy. In animals, they may delay or reduce the extent of myocardial necrosis after coronary occlusion or coronary occlusion followed by reperfusion, and in low doses that do not alter the hemodynamic profile, they have been shown to enhance the return of ventricular function in animals with stunned myocardium. However, the early first-generation calcium antagonists (nifedipine, verapamil, diltiazem) have not been shown to reduce myocardial infarct size or to enhance survival in patients with acute myocardial infarction. There now are clinical studies that suggest that, unlike beta blockers or nitrates, nifedipine may slow the development of atherosclerotic progression in humans over a 2-year period, and it seems likely that in the 1990s there will be further expansion of the use of calcium antagonists for not only angina and hypertension but also for aspects of cardioprotection. That calcium antagonists may delay, prevent or possibly regress atherosclerotic lesions is an exciting possibility.

Publication Types:
- Review

PMID: 2147358 [PubMed - indexed for MEDLINE]

1: Postgrad Med J. 1991;67 Suppl 5:S41-3.

Vascular and myocardial effects of amlodipine: an overview.

Nayler WG, Gu XH.

Department of Medicine, University of Melbourne, Austin Hospital, Heidelberg, Victoria, Australia.

Amlodipine is a long-acting dihydropyridine calcium antagonist with vascular selectivity. Although structurally related to nifedipine, amlodipine differs in several important respects, including its slow rate of onset and slow recovery. These effects probably reflect the relatively slow rate of association and dissociation of amlodipine with its binding site. The interaction of amlodipine with the calcium antagonist binding site associated with the slow Ca2+ channels differs from that of other dihydropyridines in that it involves the binding domains for the phenylalkylamine- and benzothiazepine-based antagonists, as well as for the dihydropyridines. The prolonged duration of action of amlodipine makes it suitable for use in conditions where calcium channel blockade is required on a 24-h basis. To determine whether amlodipine has a vascular protective effect, amlodipine was given orally to either cholesterol-fed rabbits or stroke-prone hypertensive rats. In the cholesterol-fed rabbits amlodipine (1 or 5 mg/kg/day) produced a significant, dose-dependent reduction in the incidence of atheromatous lesions in the thoracic aorta over an 8-week period. In stroke-prone rats the administration of amlodipine at a dose of 5 mg/kg/day reduced the incidence of mortality over a 30-week treatment period. In spontaneously hypertensive rats amlodipine (5 mg/kg/day) caused a fall in systolic blood pressure, accompanied by a significant (P less than 0.01) reduction in cardiac hypertrophy. When administered intravenously (0.25 mg/kg) 5 h before hearts were excised and made globally ischaemic for short periods (the 'stunned' heart) amlodipine pretreatment improved functional recovery associated with reperfusion.

PMID: 1839439 [PubMed - indexed for MEDLINE]

1: J Hum Hypertens. 1992 Dec;6 Suppl 1:S13-8.

Protective effects of various calcium antagonists against experimental arteriosclerosis.

Fleckenstein-Grun G, Frey M, Thimm F, Fleckenstein A.

Study Group for Calcium Antagonism, University of Freiburg, Germany.

Arterial walls altered by sclerotic processes accumulate lipids (particularly cholesterol) and calcium. Whereas the accumulation of lipids has long been incriminated as the major pathogenic factor involved in arteriosclerosis, concomitant arterial calcium overload has been considered of secondary importance. Using various animal models and specific calcium antagonists as experimental tools, we have shown the crucial role of excessive calcium uptake into arterial walls in the pathogenesis of arteriosclerotic lesions. Anticalcinotic vasoprotection with calcium antagonists has been demonstrated using light and electron microscopy, radiocalcium uptake experiments and calcium analyses with atomic absorption spectroscopy. The new 1,4-dihydropyridine calcium antagonist amlodipine has been shown to inhibit calcium accumulation in the internal elastic membrane of abdominal arteries of NaCl-loaded salt-sensitive Dahl-S rats, and consequently also exerts protective effects against arteriosclerotic lesions, shown particularly in the distal mesenteric artery branches. Formation of human coronary plaques is marked by a substantial local uptake of calcium, whereas there is a large overlap in the mural cholesterol content of healthy coronary arteries and plaques. Experimental findings in animals and with human tissue indicate that calcium antagonists such as amlodipine may provide a new approach to the prophylaxis of coronary artery lesions.

Publication Types:
- Review
- Review, Tutorial

PMID: 1293304 [PubMed - indexed for MEDLINE]

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1: Parody TR, Stone MJ. Related Articles, Links

High level expression, activation, and antagonism of CC chemokine receptors CCR2 and CCR3 in Chinese hamster ovary cells.
Cytokine. 2004 Jul 7;27(1):38-46.
PMID: 15207250 [PubMed - indexed for MEDLINE]

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Frade JM, Mellado M, del Real G, Gutierrez-Ramos JC, Lind P, Martinez-A C.

Department of Immunology and Oncology, Centro Nacional de Biotecnologia, Consejo Superior de Investigaciones Cientificas, Campus Cantoblanco, Universidad Autonoma, Madrid, Spain.

We have derived anti-human CCR2-specific mAbs by immunization with synthetic peptides corresponding to CCR2 sequences presumably involved in the interaction with its ligand(s). The characterization of these mAbs includes the ability to recognize the CCR2 receptor specifically, as well as the function based on their ability to promote Ca2+ influx or to block MCP-1-induced Ca2+ influx and chemotaxis. One mAb (MCP-1 R02) that is directed to the NH2 terminal domain of the CCR2 receptor has MCP-1 agonist activity, and two that recognize the third extracellular domain (MCP-1R04 and MCP-1 R05) have MCP-1 antagonist activity. We analyzed the presence of CCR2 in several PBL and tonsil-derived leukocyte populations and found expression of this receptor in monocytes, activated T cells, and, surprisingly, in B cells. CCR2 receptor expression in B cells was further corroborated in Southern blot using CCR2-specific probes. Moreover, both MCP-1 and the agonist mAb trigger specific B cell migration via a PTX-sensitive mechanism, indicating the presence of a functional CCR2 receptor in these cells.

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Ogilvie P, Bardi G, Clark-Lewis I, Baggiolini M, Uguccioni M.

Institute for Research in Biomedicine, Bellinzona, Switzerland.

Eotaxin is a potent inducer of eosinophil chemotaxis and was considered as a selective ligand of the CC chemokine receptor 3 (CCR3), which is expressed on eosinophils, basophils, and Th2 lymphocytes. This study shows that eotaxin also interacts with CCR2 and CCR5 and can, thus, affect the responses of monocytes, which express both receptors. In human monocytes pretreatment with eotaxin decreased responsiveness to MCP-1, a selective ligand for CCR2, as well as to RANTES and MIP-1 beta, which bind to CCR5. Similar effects were obtained with transfected cells expressing CCR2 or CCR5, but here a difference became apparent: Eotaxin triggered CCR5 at a concentration of 100 nM but not CCR2 even at 1 microM, suggesting an antagonistic effect on this receptor. In agreement with this observation, eotaxin induced internalization of CCR5 but not of CCR2 in human monocytes and transfected cells. Binding studies showed that eotaxin displaces (125) I-MCP-1 from monocytes in a concentration-dependent manner, and functional experiments showed that eotaxin inhibits MCP-1-induced chemotaxis and enzyme release. The results demonstrate that eotaxin is a CCR5 agonist and a CCR2 antagonist. The present findings suggest a role of eotaxin in the fine-tuning of cellular responses occurring at sites of allergic inflammation, in which both MCP-1 and eotaxin are produced. (Blood. 2001;97:1920-1924)

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The generation and characterisation of antagonist RNA aptamers to MCP-1.

Rhodes A, Smithers N, Chapman T, Parsons S, Rees S.

Molecular Discovery Department, Glaxo Wellcome Research and Development, Stevenage, Herts SG1 2NY, UK. adr7003@gsk.com

Monocyte chemoattractant protein-1 (MCP-1) has been implicated as a powerful pro-inflammatory mediator and may represent a potentially important, therapeutic opportunity for treatment of inflammatory disease and atherosclerosis. To further investigate the role of MCP-1 in inflammatory disorders we have isolated a series of RNA aptamers that bind specifically to mouse MCP-1. The highest affinity aptamers, designated ADR7 and ADR22, have been functionally characterised in vitro and in cell based assays. ADR7 and ADR22 have an affinity of 180 pM and 370 pM respectively for mouse MCP-1, they can antagonise MCP-1 binding to heparin and specifically antagonise MCP-1 induced chemotaxis in a cell based assay. An interesting feature of ADR22 but not ADR7 is that it is capable of antagonising the function of human MCP-1, demonstrating the high level of specificity of these aptamers and that the aptamers recognise MCP-1 in different ways. The aptamers may be used as a tool to further investigate the role of MCP-1 in inflammatory disorders and may also have a role as a therapeutic agent.

PMID: 11591377 [PubMed - indexed for MEDLINE]

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1: Blood. 2003 Aug 1;102(3):789-94. Epub 2003 Apr 10.

Eotaxin-3 is a natural antagonist for CCR2 and exerts a repulsive effect on human monocytes.

Ogilvie P, Paoletti S, Clark-Lewis I, Uguccioni M.

Institute for Research in Biomedicine, Via Vela 6, 6500 Bellinzona, Switzerland.

Eotaxin-3 (CCL26) belongs to the group of CC chemokines that attract eosinophils, basophils, and Th2 lymphocytes. Like eotaxin (CCL11) and eotaxin-2 (CCL24), eotaxin-3 mediates its activity through CCR3. Here we show that eotaxin-3 also binds to CCR2 on monocytes and CCR2-transfected cells. In contrast to monocyte chemotactic protein 1 (MCP-1; CCL2), eotaxin-3 does not trigger intracellular calcium mobilization, enzyme release, or phosphorylation of the mitogen-activated protein (MAP) kinase ERK and induces a weak chemotaxis in monocytes. Instead, eotaxin-3 inhibits MCP-1-mediated responses, thus acting as a natural antagonist for CCR2. This study also demonstrates that eotaxin-3 promotes active movement of monocytes away from a gradient of eotaxin-3 in vitro. This repellent effect is amplified when an additional gradient of MCP-1 is applied, demonstrating that the 2 mechanisms are synergistic. Eotaxin-3 effects on monocytes are largely abolished when cells are pretreated with MCP-1 or CCR2 antagonists. Like MCP-1-mediated migration, repulsion is sensitive to Bordetella pertussis toxin, indicating the involvement of Gi protein-coupled receptors. However, using transfected cells expressing CCR2 we could not detect F-actin formation or an active movement away induced by eotaxin-3, suggesting that either expression of a single receptor type is not sufficient to mediate cell repulsion or that the used transfected cell lines lack additional interaction molecules that are required for reverse migration. Eotaxin-3 was expressed by vascular endothelial cells and was essential for endothelial transmigration of eosinophils. Our data provide a mechanism by which 2 chemokine gradients that are oriented in opposite directions could cooperate in efficiently driving out monocytes from blood vessels into tissue.

PMID: 12689946 [PubMed - indexed for MEDLINE]

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1: J Med Chem. 2003 Sep 11;46(19):4070-86.

ACS PUBLICATIONS

CCR2: characterization of the antagonist binding site from a combined receptor modeling/mutagenesis approach.

Berkhout TA, Blaney FE, Bridges AM, Cooper DG, Forbes IT, Gribble AD, Groot PH, Hardy A, Ife RJ, Kaur R, Moores KE, Shillito H, Willetts J, Witherington J.

Department of Vascular Biology, GlaxoSmithKline, New Frontiers Science Park, Third Avenue, Harlow, Essex, UK CM19 5AD.

We describe here a classical molecular modeling exercise that was carried out to provide a basis for the design of novel antagonist ligands of the CCR2 receptor. Using a theoretical model of the CCR2 receptor, docking studies were carried out to define plausible binding modes for the various known antagonist ligands, including our own series of indole piperidine compounds. On the basis of these results, a number of site-directed mutations (SDM) were designed that were intended to verify the proposed docking models. From these it was clear that further refinements would be necessary in the model. This was aided by the publication of a crystal structure of bovine rhodopsin, and a new receptor model was built by homology to this structure. This latest model enabled us to define ligand-docking hypotheses that were in complete agreement with the results of the SDM experiments.

PMID: 12954060 [PubMed - indexed for MEDLINE]

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1: Am J Physiol Heart Circ Physiol. 2004 Feb;286(2):H768-74. Epub 2003 Oct 30.

Novel anti-inflammatory actions of amlodipine in a rat model of arteriosclerosis induced by long-term inhibition of nitric oxide synthesis.

Kataoka C, Egashira K, Ishibashi M, Inoue S, Ni W, Hiasa K, Kitamoto S, Usui M, Takeshita A.

Department of Cardiovascular Medicine, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.

Amlodipine (a new class of calcium channel antagonist) has been shown to limit the progression of arteriosclerosis and decrease the incidence of cardiovascular events. The mechanisms underlying the beneficial effects of amlodipine, however, remain unclear. Therefore, we hypothesized that amlodipine attenuates the development of arteriosclerosis through the inhibition of inflammation in vivo. Long-term inhibition of nitric oxide (NO) by administration of a NO synthase inhibitor, N(omega)-nitro-L-arginine methyl ester (L-NAME), to rats induces coronary vascular inflammation [monocyte infiltration, monocyte chemoattractant protein-1 (MCP-1) expression, increased activity of angiotensin-converting enzyme (ACE)], and arteriosclerosis. Here, we used the rat model to investigate the anti-inflammatory effects of amlodipine in vivo. Treatment with amlodipine markedly inhibited the L-NAME-induced increase in vascular inflammation, oxidative stress, and local ACE and Rho activity and prevented arteriosclerosis. Interestingly, amlodipine prevented the L-NAME-induced increase in MCP-1 receptor CCR2 expression in circulating monocytes. Amlodipine markedly attenuated the high mortality rate at 8 wk of treatment. These data suggest that amlodipine attenuated arteriosclerosis through inhibiting inflammatory disorders in the rat model of long-term inhibition of NO synthesis. The anti-inflammatory effects of amlodipine seem to be mediated not only by the inhibition of local factors such as MCP-1 but also by the decrease in CCR2 in circulating monocytes. Inhibition of the MCP-1 to CCR2 pathway may represent novel anti-inflammatory actions of amlodipine beyond blood pressure lowering.

PMID: 14592942 [PubMed - indexed for MEDLINE]

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1: Cytokine. 2004 Jul 7;27(1):38-46.

High level expression, activation, and antagonism of CC chemokine receptors CCR2 and CCR3 in Chinese hamster ovary cells.

Parody TR, Stone MJ.

Department of Chemistry, Indiana University, Bloomington, IN 47405-0001, USA.

The specificity of leukocyte trafficking in inflammation is controlled by the interactions of chemokines with chemokine receptors. Reliable structure-function studies of chemokine-receptor interactions would benefit from cell lines that express consistent high levels of chemokine receptors. We describe herein two new Chinese hamster ovary (CHO) cell lines in which the genes for chemokine receptors CCR2 and CCR3 have been incorporated into identical positions in the host genome. CCR2 is the primary receptor for the chemokine monocyte chemoattractant protein-1 (MCP-1) whereas CCR3 is the primary receptor for the chemokines eotaxin-1, eotaxin-2 and eotaxin-3. Both receptors are expressed at >5,000,000 copies per cell, substantially higher levels than in previous cell lines, and both are competent for binding and activation by the cognate chemokines for these receptors. Using these cell lines we confirm that eotaxin-1 and eotaxin-3 can act as an agonist and an antagonist, respectively, of CCR2. In addition, we show that eotaxin-2 is an antagonist of CCR2 and MCP-1 is an agonist of CCR3. Comparison of the chemokine sequences reveals several positions that are identical in MCP-1 and eotaxin-1 but different in eotaxin-2 and eotaxin-3, suggesting that these amino acids play a role in CCR2 activation.

PMID: 15207250 [PubMed - indexed for MEDLINE]

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Characterization of the CCR2 chemokine receptor: functional CCR2 receptor expression in B cells.

J Immunol. 1997 Dec 1;159(11):5576-84.
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Amino-terminally modified RANTES analogues demonstrate differential effects on RANTES receptors.

Proudfoot AE, Buser R, Borlat F, Alouani S, Soler D, Offord RE, Schroder JM, Power CA, Wells TN.

Serono Pharmaceutical Research Institute, 14 Chemin des Aulx, 1228 Plan-les-Ouates, Geneva, Switzerland. amanda.proudfoot@serono.com

Modification of the amino terminus of regulated on activated normal T-cell expressed (RANTES) has been shown to have a significant effect on biological activity and produces proteins with antagonist properties. Two amino-terminally modified RANTES proteins, Met-RANTES and aminooxypentane-RANTES (AOP-RANTES), exhibit differential inhibitory properties on both monocyte and eosinophil chemotaxis. We have investigated their binding properties as well as their ability to activate the RANTES receptors CCR1, CCR3, and CCR5 in cell lines overexpressing these receptors. We show that Met-RANTES has weak activity in eliciting a calcium response in Chinese hamster ovary cells expressing CCR1, CCR3, and CCR5, whereas AOP-RANTES has full agonist activity on CCR5 but is less effective on CCR3 and CCR1. Their ability to induce chemotaxis of the murine pre-B lymphoma cell line, L1.2, transfected with the same receptors, consolidates these results. Monocytes have detectable mRNA for CCR1, CCR2, CCR3, CCR4, and CCR5, and they respond to the ligands for these receptors in chemotaxis but not always in calcium mobilization. AOP-RANTES does not induce calcium mobilization in circulating monocytes but is able to do so as these cells acquire the macrophage phenotype, which coincides with a concomitant up-regulation of CCR5. We have also tested the ability of both modified proteins to induce chemotaxis of freshly isolated monocytes and eosinophils. Cells from most donors do not respond, but occasionally cells from a particular donor do respond, particularly to AOP-RANTES. We therefore hypothesize that the occasional activity of AOP-RANTES to induce leukocyte chemotaxis is due to donor to donor variation of receptor expression.

PMID: 10542293 [PubMed - indexed for MEDLINE]

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Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.
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J Biochem (Tokyo). 2001 Apr;129(4):577-83.
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2: Iyonaga K, Takeya M, Saita N, Sakamoto O, Yoshimura T, Ando M, Takahashi K. Related Articles, Links

Monocyte chemoattractant protein-1 in idiopathic pulmonary fibrosis and other interstitial lung diseases.
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Increased expression of the monocyte chemoattractant protein-1 in bronchial tissue from asthmatic subjects.
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1: Am J Respir Cell Mol Biol. 1994 Feb;10(2):142-7.

Increased expression of the monocyte chemoattractant protein-1 in bronchial tissue from asthmatic subjects.

Sousa AR, Lane SJ, Nakhosteen JA, Yoshimura T, Lee TH, Poston RN.

Department of Experimental Pathology, U.M.D.S., Guy's Hospital, London, United Kingdom.

The expression of the monocyte chemoattractant protein (MCP-1), a member of the chemokine family of low molecular weight cytokines, was assessed by immunohistochemistry in bronchial biopsies from 12 asthmatic and 12 normal subjects. Both a monoclonal antibody (F9) and a polyclonal antibody were employed to detect MCP-1, while the mouse myeloma protein (MOPC21) was used as a negative control. Strong positive reactions for MCP-1 were seen in the bronchial epithelium. Subepithelial macrophages, blood vessels, and bronchial smooth muscle were also stained. Hue-saturation-intensity color image analysis was used to quantify reactions of the monoclonal antibody in the epithelial and subepithelial layers. With the monoclonal antibody, asthmatic biopsies showed 51.8 +/- 3.7% (mean +/- SEM) of the epithelium staining positively, whereas normal subjects reacted much less, with 6.4 +/- 1.9% of the epithelium staining ($P < 0.0001$); there was no overlap between the two groups. Likewise, staining was increased in the subepithelium of asthmatic airway biopsies, with 11.5 +/- 3.1% and 2.0 +/- 1.0% staining positively in asthmatic and normal subepithelium, respectively, ($P < 0.002$). There was a significant correlation between staining of the epithelium and subepithelium ($r = 0.77$, $P < 0.001$). The polyclonal anti-MCP-1 antibody also gave strong reactions in the epithelium and subepithelium, with 34.0 +/- 7.8% of the asthmatic and 1.6 +/- 1.0% of the normal bronchial epithelium staining positively ($P < 0.0001$). These increased levels of MCP-1 in the asthmatic airways suggest that they may play a role in macrophage recruitment and activation and thereby contribute to the inflammatory pathology of bronchial asthma.

PMID: 8110469 [PubMed - indexed for MEDLINE]

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Monocyte chemoattractant protein-1 in idiopathic pulmonary fibrosis and other interstitial lung diseases.

Iyonaga K, Takeya M, Saita N, Sakamoto O, Yoshimura T, Ando M, Takahashi K.

Second Department of Pathology, Kumamoto University School of Medicine, Japan.

Macrophages play a crucial role in the pathogenesis of idiopathic pulmonary fibrosis (IPF). To examine the mechanisms for increased monocyte/macrophage recruitment in IPF and nonIPF interstitial lung diseases (nonIPF) the localization of monocyte chemoattractant protein-1 (MCP-1) was investigated in 14 cases of IPF, seven cases of nonIPF, and seven normal control lungs (CTRL) by immunohistochemistry using a specific anti-MCP-1 monoclonal antibody, F9. By double immunohistochemical staining using F9 and one of the cell type specific antibodies significant differences in the staining pattern of MCP-1 were observed between IPF and nonIPF. In IPF MCP-1 was observed in cuboidal and flattened metaplastic epithelial cells, alveolar macrophages, and vascular endothelial cells. In contrast, no epithelial cells were stained for MCP-1 in nonIPF cases, although alveolar macrophages and vascular endothelial cells were labeled. Northern hybridization analysis of selected cases showed marked expression of MCP-1 messenger RNA (mRNA) in IPF and nonIPF compared with CTRL. These findings suggest that the MCP-1 production in IPF and nonIPF plays an important role in the recruitment of monocyte/macrophages. Monocyte chemoattractant protein-1 production by epithelial cells in IPF may be caused by the metaplastic nature of the epithelial cells and may be one of the key factors inducing the irreversible progression of IPF.

PMID: 8200639 [PubMed - indexed for MEDLINE]

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Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

In this study, we attempted to analyze the peptide motifs recognized by 24822.111 and F9, monoclonal antibodies (mAbs) that inhibit the chemotactic activity of monocyte chemoattractant protein-1 (MCP-1), a member of the CC subfamily of chemokines. We isolated phage clones from a phage display library and identified six peptide motifs. One of these clones, C27, was strongly and specifically recognized by 24822.111 mAb, while another, G25, was similarly recognized by F9 mAb. Both the C27 motif and the G25 motif contain two cysteines in their sequences and have little homology to the primary amino acid sequence of MCP-1. These clones, however, bound to THP-1 cells, and the binding was competitively inhibited by MCP-1. The clones strongly inhibited the MCP-1-induced chemotaxis of human monocytes. The synthetic and intramolecularly disulfide-linked peptides of C27 and G25 (sC27 and sG25) also inhibited the chemotaxis induced by MCP-1, while their derivatives with serine in place of cysteine did not, suggesting the importance of the loop structure for the inhibition. These results suggest that sC27 and sG25 may mimic the MCP-1-binding domain to the MCP-1 receptor.

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1: J Hum Hypertens. 1992 Dec;6 Suppl 1:S19-23.

The antiatherogenic effects of amlodipine: promise of preclinical data.

Nayler WG.

Department of Medicine, University of Melbourne, Austin Hospital, Heidelberg, Victoria, Australia.

Atherosclerosis is a complex and multifactorial disease, the endpoint of which is the formation of a calcified plaque. Intermediate events include intimal injury, smooth muscle cell proliferation and migration, macrophage infiltration, lipid accumulation and excess formation of ground substance. To determine whether the newly developed, long-acting calcium antagonist, amlodipine, slows the development of atherosclerotic lesions under experimental conditions, young New Zealand white rabbits were fed on a diet of 2% cholesterol plus 1% peanut oil for up to 12 weeks. Half the rabbits received 1 or 5 mg amlodipine/kg body weight/day. Amlodipine caused a significant and dose-dependent reduction in lesion formation in the thoracic aorta. At the same time thoracic aorta Ca2+ and cholesterol content were maintained at near normal levels, despite the raised plasma cholesterol levels. The protective effect of amlodipine persisted throughout a treatment period of 12 weeks, indicating the absence of tachyphylaxis.

PMID: 1284082 [PubMed - indexed for MEDLINE]

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Propagermanium
Antibody 24822.111
Antibody F9
Eotaxin: eotaxin-2, eotaxin-3 but NOT Eotaxin-1
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RNA ADR22
Eotaxin-3
Amlodipine

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AN    2004265315   BIOENG
DN    3726839
TI      ***Antagonists***   of monocyte chemoattractant protein 1 identified by
      modification of functionally critical NH sub(2)-terminal residues
AU    Gong, Jiang-Hong; Clark-Lewis, I*
CS    Biomed. Res. Cent., 2222 Health Sci. Mall, Univ. British Columbia,
      Vancouver, BC V6T 1Z3, Canada
SO    Journal of Experimental Medicine [J. EXP. MED.], vol. 181, no. 2, pp.
      631-640, 1995
      ISSN: 0022-1007
DT    Journal
LA    English
SL    English
OS    Immunology Abstracts; Chemoreception Abstracts

L4   ANSWER 2 OF 13  CANCERLIT on STN
AN   2002020966     CANCERLIT
DN   95279804   PubMed ID: 7759884
TI   Monocyte chemoattractant protein-3, but not monocyte chemoattractant
     protein-2, is a functional ligand of the human monocyte chemoattractant
     protein-1 receptor.
```

AU Franci C; Wong L M; Van Damme J; Proost P; Charo I F
CS Gladstone Institute of Cardiovascular Disease, San Francisco, CA 94110, USA.
NC HL52773 (NHLBI)
SO JOURNAL OF IMMUNOLOGY, ***(1995 Jun 15)*** 154 (12) 6511-7.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Abridged Index Medicus Journals; Priority Journals
OS MEDLINE 95279804
EM 199506
ED Entered STN: 20020726
Last Updated on STN: 20020726

L4 ANSWER 3 OF 13 CANCERLIT on STN
AN 95197599 CANCERLIT
DN 95197599 PubMed ID: 7890708
TI Signal transduction and ligand specificity of the human monocyte chemoattractant protein-1 receptor in transfected embryonic kidney cells.
AU Myers S J; Wong L M; Charo I F
CS Gladstone Institute of Cardiovascular Disease, San Francisco, California 94141-9100.
NC HL52773 (NHLBI)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, ***(1995 Mar 17)*** 270 (11) 5786-92.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 95197599
EM 199504
ED Entered STN: 19950509
Last Updated on STN: 19970509

L4 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:856120 CAPLUS
DN 123:254582
TI Mammalian monocyte chemoattractant protein receptors and cDNAs encoding them and their therapeutic use
IN Charo, Israel; Coughlin, Shaun
PA Regents of the University of California, USA
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9519436	A1	19950720	WO 1995-US476	19950111 <--
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US				
	RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9516793	A1	19950801	AU 1995-16793	19950111 <--
	EP 740701	A1	19961106	EP 1995-908495	19950111
	R: AT, CH, DE, FR, GB, LI, SE				
	JP 09508264	T2	19970826	JP 1995-519133	19950111
	JP 3532570	B2	20040531		
	US 6132987	A	20001017	US 1995-446669	19950525
	US 6730301	B1	20040504	US 2000-625573	20000725
	US 2004219644	A1	20041104	US 2004-791592	20040301
	US 2004223968	A1	20041111	US 2004-791166	20040301
PRAI	US 1994-182962	A	19940113		
	US 1995-446669	A1	19950111		
	WO 1995-US476	W	19950111		
	US 2000-625573	A1	20000725		

L4 ANSWER 5 OF 13 CEABA-VTB COPYRIGHT 2005 DECHEMA on STN
AN 1996(06):6425 CEABA-VTB FS B
DN CEABA: 1996:2291891
TI Mammalian monocyte chemoattractant protein receptors
AU Charo, I. (Univ. California, Oakland, CA 94612-3550, USA)

SO PCT Patent Appl. (***1995***) WO 9519436(Appl. US 08/182962 Filed 13 Jan 1994)
CODEN: PIXXD2
DT Patent
LA English

L4 ANSWER 6 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAR79165 Protein DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic. for identifying ***antagonists*** and for treating diseases characterised by monocytic infiltrates
IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
CR N-PSDB: AAQ96297
DESC Human monocyte chemoattractant protein-1 receptor ***MCP*** - ***1RA*** .

L4 ANSWER 7 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAR79170 Peptide DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic. for identifying ***antagonists*** and for treating diseases characterised by monocytic infiltrates
IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
DESC End of third transmembrane domain of human MIP-1alpha/RANTES receptor.

L4 ANSWER 8 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAR79169 Peptide DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic. for identifying ***antagonists*** and for treating diseases characterised by monocytic infiltrates
IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
DESC End of third transmembrane domain of human monocyte chemoattractant protein-1 receptor MCR-1RA.

L4 ANSWER 9 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAR79166 Protein DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic. for identifying ***antagonists*** and for treating diseases characterised by monocytic infiltrates
IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
CR N-PSDB: AAQ96298
DESC Human monocyte chemoattractant protein-1 receptor ***MCP*** - ***1RB*** .

L4 ANSWER 10 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAQ96297 cDNA DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic. for identifying ***antagonists*** and for treating diseases characterised by monocytic infiltrates

IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A1 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
CR P-PSDB: AAR79165
DESC Human monocyte chemoattractant protein-1 receptor ***MCP*** -
  1RA .

L4 ANSWER 11 OF 13 DGENE COPYRIGHT 2005 The Thomson Corp on STN
AN AAQ96298 cDNA DGENE
TI DNA encoding monocyte chemo-attractant protein-1 receptor - used partic.
  for identifying ***antagonists*** and for treating diseases
  characterised by monocytic infiltrates
IN Charo I; Coughlin S
PA (REGC) UNIV CALIFORNIA.
PI ***WO 9519436 A1 19950720 84p***
AI WO 1995-US476 19950111
PRAI US 1994-182962 19940113
DT Patent
LA English
OS 1995-263866 [34]
CR P-PSDB: AAR79166
DESC Human monocyte chemoattractant protein-1 receptor ***MCP*** -
  1RB .

L4 ANSWER 12 OF 13 IMSDRUGNEWS COPYRIGHT 2005 IMSWORLD on STN

ACCESSION NUMBER: 94:622 IMSDRUGNEWS
TITLE: Novel Approaches to Inflammation Intervention at LeukoSite
SOURCE: R&D Focus Drug News (***13 Jun 1994***).
WORD COUNT: 504

L4 ANSWER 13 OF 13 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
AN 1993-145687 [18] WPIDS
DNC C1993-064992
TI 5-Bi phenylyl tetrazole cpd. prepn. for angiotensin ***antagonist***
  intermediate - by reacting fluorophenyl tetrazole with Grignard reagent,
  for high yield avoiding utilisation or generation of highly toxic
  materials e.g. tin reagents.
DC B03
IN MURRAY, W V; RUSSELL, R
PA (ORTH) ORTHO PHARM CORP
CYC 19
PI EP 540356 A2 19930505 (199318)* EN 8 C07D257-04 <--
    R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE
  AU 9227404 A 19930506 (199325) C07D257-04 <--
  CA 2081847 A 19930502 (199329) C07D257-04 <--
  US 5252753 A 19931012 (199342) 5 C07D257-04 <--
  JP 05279350 A 19931026 (199347) 7 C07D257-04 <--
  AU 651014 B 19940707 (199431) C07D257-04 <--
  EP 540356 A3 19930825 (199508) C07D257-04 <--
  SG 47012 A1 19980320 (199818) C07D257-04
  EP 540356 B1 19990324 (199916) EN C07D257-04
    R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE
  DE 69228727 E 19990429 (199923) C07D257-04
  ES 2130161 T3 19990701 (199933) C07D257-04
  JP 3145813 B2 20010312 (200116) 8 C07D257-04
ADT EP 540356 A2 EP 1992-309968 19921030; AU 9227404 A AU 1992-27404 19921028;
  CA 2081847 A CA 1992-2081847 19921030; US 5252753 A US 1991-786666
  19911101; JP 05279350 A JP 1992-315657 19921030; AU 651014 B AU 1992-27404
  19921028; EP 540356 A3 EP 1992-309968 19921030; SG 47012 A1 SG 1996-1913
  19921030; EP 540356 B1 EP 1992-309968 19921030; DE 69228727 E DE
  1992-628727 19921030, EP 1992-309968 19921030; ES 2130161 T3 EP
  1992-309968 19921030; JP 3145813 B2 JP 1992-315657 19921030
FDT AU 651014 B Previous Publ. AU 9227404; DE 69228727 E Based on EP 540356;
  ES 2130161 T3 Based on EP 540356; JP 3145813 B2 Previous Publ. JP 05279350
PRAI US 1991-786666 19911101
IC ICM C07D257-04
  ICS C07D405-10; C07F007-10
ICA A61K031-41; A61K031-695; C07B047-00; C07F003-02

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=> S eotaxin OR amlodipine OR propagermanium
  27 FILES SEARCHED...
  46 FILES SEARCHED...
  48 FILES SEARCHED...
L5       43336 EOTAXIN OR AMLODIPINE OR PROPAGERMANIUM

=> S rheumatoid arthritis OR alvcolitis OR artherosclerosis
  17 FILES SEARCHED...
  28 FILES SEARCHED...
  49 FILES SEARCHED...
  65 FILES SEARCHED...
L6      973475 RHEUMATOID ARTHRITIS OR ALVCOLITIS OR ARTHEROSCLEROSIS

=> S L5 AND L6
  30 FILES SEARCHED...
L7         1265 L5 AND L6

=> DUP REM Ly
ENTER L# LIST OR (END):L7
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGMONOG2, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, IMSRESEARCH, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
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referenced by any of the L#s specified for this DUPLICATE command.
The file names of duplicates that can be kept are listed above.
Please enter one of these file names.

=> DUP REM L7
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGMONOG2, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, IMSRESEARCH, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
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PROCESSING COMPLETED FOR L7
L8          1093 DUP REM L7 (172 DUPLICATES REMOVED)

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  44 FILES SEARCHED...
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  60 FILES SEARCHED...
  65 FILES SEARCHED...
  71 FILES SEARCHED...
L9           24 L8 AND PY<=1995

=> D L9 1-24

L9   ANSWER 1 OF 24  BIOBUSINESS  COPYRIGHT (c) 1998 The Thomson Corporation.
     on STN
AN   95:87534  BIOBUSINESS
DN   0757939
TI   Pfizer Norvasc "PRAISE II" morbidity/mortality trial in non-ischemic CHF
     under way; Tenidap data on slowing bone erosion in   ***rheumatoid***
       ***arthritis***   filed.
AU   Anon
SO   F-D-C Reports - "The Pink Sheet", (  ***1995***  ) Vol.57, No.44, Oct. 30,
     P.12-13.
     ISSN: 1068-5324.
FS   UNIQUE
LA   ENGLISH
```

L9 ANSWER 2 OF 24 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
AN 1993:23096738 BIOTECHNO
TI FDA clears 26 new drugs for marketing in 1992
SO American Journal of Hospital Pharmacy, (***1993***), 50/4 (578+581)
CODEN: AJHPA0 ISSN: 0002-9289
DT Journal; Note
CY United States
LA English

L9 ANSWER 3 OF 24 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 94099138 EMBASE
DN 1994099138
TI [Cyclosporin-A in refractory ***rheumatoid*** ***arthritis***].
TRAITEMENT DE LA POLYARTHRITE RHUMATOIDE REFRACTAIRE PAR LA CICLOSPORINE.
AU Flipo R.M.; Cortet B.; Duquesnoy B.; Delcambre B.
CS Service de Rhumatology, Hopital B, CHRU,59037 Lille Cedex, France
SO Revue de Medecine Interne, (1994) 15/3 (166-173).
ISSN: 0248-8663 CODEN: RMEIDE
CY France
DT Journal; Article
FS 006 Internal Medicine
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LA French
SL French; English

L9 ANSWER 4 OF 24 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 93120951 EMBASE
DN 1993120951
TI 1992 drug approvals: The year in review.
AU Estrada J.
SO Drug Therapy, (1993) 23/3 (55-58+63).
ISSN: 0001-7094 CODEN: DRTHE2
CY United States
DT Journal; Note
FS 030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English

L9 ANSWER 5 OF 24 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 93096738 EMBASE
DN 1993096738
TI FDA clears 26 new drugs for marketing in 1992.
SO American Journal of Hospital Pharmacy, (1993) 50/4 (578+581).
ISSN: 0002-9289 CODEN: AJHPA
CY United States
DT Journal; Note
FS 004 Microbiology
006 Internal Medicine
016 Cancer
030 Pharmacology
037 Drug Literature Index
LA English

L9 ANSWER 6 OF 24 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 91286998 EMBASE
DN 1991286998
TI [Regression of coronary ***artherosclerosis*** : Is it feasible and is
it relevant?].
REGRESSION DER KORONARATHEROMATOSE: MOGLICH - UND VON PRAKTISCHER
BEDEUTUNG?.
AU Niederhauser H.U.
CS Klinik, 9056 Gais, Switzerland
SO Schweizerische Rundschau fur Medizin/Praxis, (1991) 80/39 (1019-1023).
ISSN: 0369-8394 CODEN: SRMPDJ
CY Switzerland
DT Journal; Article
FS 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery

037 Drug Literature Index
LA German
SL English; French

L9 ANSWER 7 OF 24 IMSDRUGNEWS COPYRIGHT 2005 IMSWORLD on STN

ACCESSION NUMBER: 93:219 IMSDRUGNEWS
TITLE: Pfizer Plans NDA Filings
SOURCE: R&D Focus Drug News (***8 Mar 1993***).
WORD COUNT: 153

L9 ANSWER 8 OF 24 PHARMAML COPYRIGHT 2005 MARKETLETTER on STN
AN 1626972 PHARMAML
TI New Chemical Entities Round-Up For 1994
SO Marketletter February 6, 1995
DT Newsletter
WC 1262

L9 ANSWER 9 OF 24 PHARMAML COPYRIGHT 2005 MARKETLETTER on STN
AN 1619078 PHARMAML
TI PFIZER "COMMITTED" TO HEALTH CARE AND R&D
SO Marketletter March 8, 1993
DT Newsletter
WC 459

L9 ANSWER 10 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 93:14786 PHIN
DN S00376488
DED 1 Oct 1993
TI Slowdown in Finnish pharmaceutical spending in 1992
SO Scrip (***1993***) No. 1860 p5
DT Newsletter
FS FULL

L9 ANSWER 11 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 93:3704 PHIN
DN S00353790
DED 9 Mar 1993
TI Pfizer reviews upcoming filings
SO Scrip (***1993***) No. 1801 p11
DT Newsletter
FS FULL

L9 ANSWER 12 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 92:11209 PHIN
DN S00318962
DED 14 Aug 1992
TI Pfizer riding high on investor confidence
SO Scrip (***1992***) No. 1744 p8
DT Newsletter
FS FULL

L9 ANSWER 13 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 92:8452 PHIN
DN S00313362
DED 19 Jun 1992
TI Pfizer's largest filing - tenidap?
SO Scrip (***1992***) No. 1728 p25
DT Newsletter
FS FULL

L9 ANSWER 14 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 91:12851 PHIN
DN S00280114
DED 19 Jul 1991
TI 329 Pharmaceutical Manufacturers Association (PMA) drugs in development
for the aging
SO Scrip (***1991***) No. 1635 p24
DT Newsletter
FS FULL

L9 ANSWER 15 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 91:8387 PHIN
DN S00278350
DED 26 Jun 1991
TI Recent drug approvals for Pfizer worldwide
SO Scrip (***1991***) No. 1628 p24
DT Newsletter
FS BRIEF

L9 ANSWER 16 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 91:1549 PHIN
DN S00262034
DED 11 Jan 1991
TI Pfizer's best Research & Development (R&D) pipeline ever
SO Scrip (***1991***) No. 1581 p12
DT Newsletter
FS FULL

L9 ANSWER 17 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 89:17085 PHIN
DN S00203198
DED 9 Jun 1989
TI Pfizer's ***amlodipine*** - 1st approval
SO Scrip (***1989***) No. 1419 p24
DT Newsletter
FS FULL

L9 ANSWER 18 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 89:957 PHIN
DN S00222444
DED 8 Dec 1989
TI Drugs for the US elderly
SO Scrip (***1989***) No. 1471 p30
DT Newsletter
FS FULL

L9 ANSWER 19 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 88:15724 PHIN
DN S00164570
DED 1 Jul 1988
TI Short-term problems for Pfizer?
SO Scrip (***1988***) No. 1324 p8
DT Newsletter
FS FULL

L9 ANSWER 20 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 88:682 PHIN
DN S00152630
DED 21 Mar 1988
TI Pfizer's new anti-inflammatory
SO Scrip (***1988***) No. 1294 p12
DT Newsletter
FS FULL

L9 ANSWER 21 OF 24 PHIN COPYRIGHT 2005 T&F Informa UK Ltd on STN

AN 86:13036 PHIN
DN S00102047
DED 10 Dec 1986
TI Products in the news in 1986
SO Scrip (***1986***) No. 1166/7 p11
DT Newsletter
FS FULL

L9 ANSWER 22 OF 24 PROMT COPYRIGHT 2005 Gale Group on STN

ACCESSION NUMBER: 93:531562 PROMT
TITLE: PFIZER "COMMITTED" TO HEALTH CARE AND R&D
SOURCE: Marketletter, (***8 Mar 1993***) pp. N/A.
ISSN: 0140-4288.

LANGUAGE: English
WORD COUNT: 450
FULL TEXT IS AVAILABLE IN THE ALL FORMAT

L9 ANSWER 23 OF 24 PROMT COPYRIGHT 2005 Gale Group on STN

ACCESSION NUMBER: 92:544296 PROMT
TITLE: Pfizer-Eleven New Products: Near Term Pipeline Impressive
SOURCE: Genesis Report-Rx, (***Dec 1991***) pp. N/A.
ISSN: 1061-2270.
LANGUAGE: English
WORD COUNT: 1355
FULL TEXT IS AVAILABLE IN THE ALL FORMAT

L9 ANSWER 24 OF 24 USPATFULL on STN
AN 95:114762 USPATFULL
TI Inhibition of arthritis by L-type calcium channel antagonists nimodipine, nisoldipine and nifedipine
IN Aune, Thomas M., Hamden, CT, United States
PA Bayer Corporation, Pittsburgh, PA, United States (U.S. corporation)
PI US 5478848 19951226 <--
AI US 1994-188464 19940126 (8)
DT Utility
FS Granted
LN.CNT 537
INCL INCLM: 514/356.000
INCLS: 514/355.000
NCL NCLM: 514/356.000
NCLS: 514/355.000
IC [6]
ICM: A61K031-44
EXF 514/355; 514/356
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
STN INTERNATIONAL LOGOFF AT 17:37:31 ON 08 FEB 2005